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Epidemiology and Prevention

Stress oxidative and progression of atherosclerosis

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Core tip: Oxidative stress is involved in the pathogenesis of various cardiovascular diseases including atherosclerosis as a fibroproliferative process. Oxidation of LDL particles by active oxygen species leads to the formation of oxLDL in the intima layer and progression of atherosclerosis. Therefore, oxidative stress has been considered as one of the main risk factors for cardiovascular disease.

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Coronary artery disease (CAD) is known as the most common causes of mortality over recent decades. Atherosclerosis is a chronic inflammatory disease which plaque deposits are formed in the arterial wall and ultimately leads to vascular obstruction and myocardial infarction. Many risk factors can be the cause of atherosclerosis, such as hypercholesterolemia, hypertension, age, gender, obesity, diabetes mellitus, cigarette smoking, and inadequate lifestyle (1). In the atherosclerosis process, hypercholesterolemia activates endothelial cells in arteries. LDL particles converted to oxidized LDL (oxLDL) in the intima layer under the influence of active oxygen species, myeloperoxidase and lipoproteinases. OxLDLs are self-stimulating inflammatory responses that result in the absorption of monocytes of blood from endothelial cells and convert them to macrophages. Then, macrophages absorb oxLDL particles through their receptors and ultimately transform into foam and apoptotic cells. A set of cholesterol and apoptotic cells create a necrotic nucleus. The necrotic nucleus is protected by smooth muscle cells, endothelial cells, and collagen called a fibrous cap that prevented from entering the bloodstream. However, fibrous cap decomposes gradually through secretion of matrix metalloproteinases (MMPs) and enters the bloodstream. Finally, the platelets are activated and the set of these events will block the vessels (2).

Generally, the oxLDLs involved in several biological pathways include enhancing the expression of growth factors and related receptors, activation of phospholipase D, inducing extracellular matrix (ECM) modification, and the sphingomyelin/ceramide lactosylceramide generation (3). Recently, oxidative stress has been considered as one of the main risk factors for cardiovascular disease, and many evidence indicates the role of oxidative stress in the progression of atherogenesis. Stress oxidative is an imbalance between the generation of reactive oxygen species (ROS) and the ability of the biological system to detoxify them or to repair the damage due to ROS (4). Reactive oxygen species play the main role in the prevalence of vascular disorders such as apoptosis, adhesion molecule expression, lipid oxidation, altered vasomotion, hypertension, diabetes, aging, hypercholesterolemia, and atherosclerosis (3). The sources of stress oxidative in the vascular wall can refer to nitric oxide synthases, NADPH oxidase, myeloperoxidase, lipoxygenase/cyclooxygenase, and xanthine oxidase (5).

Numerous studies demonstrated that oxidative stress can affect vascular function through several mechanisms. ROS, especially hydroxyl radicals, damage the cell membrane and the nucleus, directly. Another mechanism in which the oxidative stress affects atherogenesis via the production of transcription factors such as the nuclear factor kB (NF-kB) and the activating protein 1 (AP-1), which involved in the expression of adherence molecules such as vascular cell adhesion molecule 1 (VCAM-1), E Selectin and other cytokines. Additionally, it causes the peroxidation of lipid components and the formation of Ox-LDL as a key intermediary of atherosclerosis (6, 7). All of these events are associated with the accumulation of macrophages and foam cells that at the end, activates platelets and the formation of thrombosis. Therefore, oxidative stress detects to play an important role in the early and final stages of atherosclerosis (8, 9). Several evidence reveals the associated between renal disease and occurrence of CAD. For example, it has been shown that urea concentrations in chronic renal failure (CRF) patients can induce mitochondrial ROS production that stimulates expression of MCP-1, VCAM1 and increase the factors leading to atherosclerosis progression (10). The reduced glomerular filtration rate and increased albuminuria are

two main factors in the progression of cardiovascular disease (CVD). Thus, patients with glomerular filtration rate below 60 mL/min/1.73 m^2 are exposed to a higher risk of CAD, congestive heart failure, peripheral artery disease and stroke (11). NADPH oxidase is one of the enzymes that associated with overproduction of ROS. Stress oxidative induced by hemodialysis activates production of NADPH oxidase significantly in dialysis patients and increase diseases development such as atherosclerosis and cardiovascular events in long-term. A study revealed that nutritional and inflammatory markers can have a role in occurrence of cardiovascular diseases in Chronic kidney disease (CKD) patients. Additionally, the relationship between stress oxidative and malnutrition is well-known (12). Recently, the use of antioxidant to be increased in the improvement of various diseases. Considering the important role of stress oxidative in the pathogenesis of atherosclerosis, it seems that antioxidant therapy can be useful in improvment and treatment of atherosclerosis. For example, it has been reported that, polyphenols, carotenoids, selenium, vitamin E, and vitamin C could be effective, although, further researches on the issue is still required (13).

Author's contribution

BY is the single author of the paper.

Conflicts of interest

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Ethical considerations

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